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The Effects of Heat Acclimatization, Heat Acclimation, and Intermittent Exercise on Aerobic
Performance

Lauren A. Fry

B.S., The Pennsylvania State University, 2018

A Thesis

Submitted in Partial Fulfillment of the
Requirements for the Degree of Master of Science

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2020

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APPROVAL PAGE

Master of Science Thesis

The Effects of Heat Acclimatization, Heat Acclimation, and Intermittent Exercise on Aerobic
Performance

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Abstract

The primary aim of this study is to determine if heat acclimation (HA)/acclimatization (HAz) improves $\text{VO}_{2\text{max}}$. A secondary aim of this study is to examine whether or not any changes in $\text{VO}_{2\text{max}}$ occur during intermittent exercise heat exposures (HAM). Twenty-seven male endurance runners (mean \pm SD: age: 36 \pm 12 years, nude body mass: 73.03 \pm 8.97 kg, height: 178.81 \pm 6.39 cm, $\text{VO}_{2\text{max}}$: 57.48 \pm 7.03 ml \cdot kg $^{-1}\cdot$ min $^{-1}$) performed five $\text{VO}_{2\text{max}}$ tests at various time points (baseline, test 1; post-HAz, test 2; post-HA, test 3; 4 weeks of HAM, test 4; 8 weeks of HAM, test 5). Participants completed a summer training regimen after test 1. After summer training, each participant completed a 5-day HA protocol. Participants were randomly assigned to one of three groups for the HAM, once a week (n=9), twice a week (n=10), or not at all/control (n=8). Differences in $\text{VO}_{2\text{max}}$, $\dot{V}\text{O}_2$, and max HR for tests 1-3 were analyzed using one-way ANOVAs while tests 3-test 5 were analyzed using repeated measure ANOVAs with Bonferroni corrections post-hoc. Statistical significance was defined as $p<0.05$, a priori. There were no significant differences in $\text{VO}_{2\text{max}}$ data between any of the $\text{VO}_{2\text{max}}$ tests 1-3 (mean \pm SD: 57.92 \pm 6.8, 59.65 \pm 8.2, 59.49 \pm 7.2 ml \cdot kg $^{-1}\cdot$ min $^{-1}$, $p=0.363$). There were no significant group or time effects for tests 3-5 ($p=0.671$), therefore no differences were found between experimental groups. There were significant differences in maximal heart rate (HR) between test 1-3 (mean \pm SD: 180 11bpm, 177 \pm 10bpm, 175 \pm 10bpm, $p=0.006$). There were significant differences in HR between test 1 and test 3 (mean \pm SD: 180 \pm 11bpm vs. 175 \pm 10bpm, $p<0.001$). There were no significant differences in HR between group or time effects for tests 3-5 ($p=0.59$). The decrease in HR may be explained as a successful induction of HA. In conclusion, there were no improvements in aerobic performance ($\text{VO}_{2\text{max}}$) following HA.

Chapter One: Literature Review

This review of the literature will provide an in-depth overview of thermoregulation, exercise in the heat, heat acclimation, and aerobic performance enhancement during heat acclimation (HA). The main focus of this chapter is to examine the current literature and supporting evidence of the effects that HA has on aerobic performance.

Thermoregulation during heat stress

Heat Balance Equation

The rate and amount of heat exchange between an individual and their environment is presented in the heat balance equation. The heat balance equation is an equation that contains both physiological and environmental factors that affect heat exchange. Core body temperature is determined by the balance between heat accumulation (metabolic heat production) and heat dissipation (Moran *et al.*, 2007; Casa *et al.*, 2015). The heat balance equation is as follows:

$$S = M(\pm W) - E \pm R \pm C \pm K$$

(Casa *et al.*, 2015)

Where S represents the stored heat, a positive S represents heat storage and a negative S represents heat loss. M represents metabolic heat production, W represents work, E represents evaporative heat loss, R represents heat lost or gained during radiation, C represents heat lost or gained during convection, and K represents heat lost or gained during conduction (Casa *et al.*, 2015). K is only important when considering direct contact with an object (Sawka *et al.*, 2011).

Radiation transfers heat using electromagnetic radiation and can include something as simple as sunlight (Casa *et al.*, 2015). Convection is the transfer of heat by the surrounding air or fluid (Casa *et al.*, 2015). Rate of convective heat loss is dictated by the thermal gradient between air and skin temperature. Conduction is a result of heat transfer by direct contact, such as hot packs applied to the skin (Casa *et al.*, 2015). Evaporation is the most efficient form of heat loss and occurs when an individual sweats and it becomes vaporized (Epstein, 1990; Casa *et al.*, 2015). It has been demonstrated that evaporative cooling of the skin reduces cardiovascular strain and reduces fatigue during heat stress (Periard *et al.*, 2016). Clothing, pads, helmets, little to no wind speed, and high humidity are all factors that will impede evaporative sweating (Cleary, 2007; Casa *et al.*, 2015). Heat dissipation can be complex and involves many body systems but it is primarily comprised of vasomotor and sudomotor activity (Moran *et al.*, 2007). Heat storage can be the result of either exorbitant heat accumulation or the reduced ability to dissipate heat (Moran *et al.*, 2007). A figure of the heat transfer mechanisms can be found in Figure 1 (Zhang & Wang, n.d.).

Insert Figure 1 here

Physiological responses to exercise in the heat

Cardiovascular, skeletomuscular, neurological systems

The body system that endures the majority of the work during heat stress is the cardiovascular system. However, when the body is put under heat stresses, the central nervous system (CNS), the cardiovascular system, and the integumentary system work simultaneously to maintain a core body temperature of approximately 37°C (Cleary, 2007; Rizzo & Thompson, 2018). The most common thermoregulatory responses that occur during heat stress are cutaneous vasodilation to

increase sweating, increased heart rate and increased respiratory rate (Cleary, 2007). The cardiovascular system acts to minimize physiological strain and better sustain exercise during heat stress by cutaneous vasodilation or increasing blood flow to the skin (Periard *et al.*, 2016). According to Lorenzo *et al.*, exercise in the heat causes skin microcirculation which enhances evaporative cooling from increased sweating (Lorenzo & Minson, 2010). This widens the core-to-skin thermal gradient further allowing heat dissipation from the core to the environment (Lorenzo & Minson, 2010).

During exercise in the heat, activated muscles produce body heat (Mee *et al.*, 2015). This heat production is an active part of the heat balance equation previously discussed. Individual variability exists in one's ability to tolerate heat stresses (Epstein, 1990). Epstein *et al.* suggests that heat accumulation is generated by either metabolic heat (physical activity) or environment heat (Epstein, 1990). Heat storage is the result of either excessive heat accumulation from either mechanism or the reduced ability of an individual to dissipate heat (Epstein, 1990). A higher heat tolerance requires a functional thermoregulatory center, cardiovascular system, and integrity of the skin and dermal appendages (Epstein, 1990).

While much of thermoregulation is attributed to the cardiovascular and skeletomuscular system, the central nervous system (CNS) plays an enormous role during exercise in the heat. In order for the body to defend itself from potentially dangerous changes in temperature, arterial oxygen tension, blood pressure, and energy depletion, the CNS uses sensory receptors to respond and maintain homeostasis (Armstrong & Stoppani, 2002). Cells of the brainstem, spinal cord, and hypothalamus work together to integrate sensory (afferent) neural signals and transmit efferent

spinal output (Armstrong & Stoppani, 2002). The afferent and efferent neural pathways of the CNS create a complex feedback loop that is responsible for automatic brain responses to pain and thermal sensation. This feedback loop is comprised of many neurons, including some warm-sensitive neurons of the spinoreticular tract that ascend into the spinal cord and directly control responses of the hypothalamus, see Figure 2 (Belval, n.d.). The preoptic anterior hypothalamus houses many of the temperature sensitive neurons and allows for the hypothalamus to initiate thermoregulatory responses (Wendt *et al.*, 2007; Sawka *et al.*, 2011; Periard *et al.*, 2015). Thermoregulatory responses occur in several phases and in different locations of the brain, all utilizing a negative feedback loop (Bongers *et al.*, 2020). A visual representation of this negative feedback loop can be found in Figure 4 (Bongers *et al.*, 2020).

Furthermore, the two primary physiological mechanisms responsible for mitigating increases in core temperature during exercise in the heat are 1) vasodilation and 2) sweating (Wendt *et al.*, 2007; Mack & Nadel, 2011; Pryor *et al.*, 2019a). These physiological responses during exercise in the heat are controlled by the principles of the heat balance equation and integration of the central nervous system.

Insert Figure 2 here

Vasodilation

Vasodilation is important for modulating skin blood flow to allow heat transfer from the core to the skin (Wendt *et al.*, 2007). During regular exercise, blood is redistributed towards working skeletal muscle tissues. This causes an increase in core temperature from metabolic heat production (Wendt *et al.*, 2007). Following a period of HA, the core temperature threshold for

the onset of cutaneous vasodilation is reduced; this can be seen in Figure 3 (Periard *et al.*, 2015). Cutaneous vasodilation is a function of the efferent pathway of the spinoreticular tract or thermoregulatory negative feedback loop (Wendt *et al.*, 2007; Bongers *et al.*, 2020). The core-to-skin thermal gradient is enhanced for heat acclimated individuals because of the increased ability of the cardiovascular system to supply the skin with microcirculation and higher sweat rates (Lorenzo & Minson, 2010).

Insert Figure 3 here

Sweating

The sweat response is one of the most important mechanisms for heat dissipation. Sweat promotes heat loss by the process of evaporation of the water content in sweat (Wendt *et al.*, 2007). Aside from water content, the main components of sweat concentration are sodium chloride, potassium, calcium and magnesium (Wendt *et al.*, 2007). Sweat concentration is modified during exercise in the heat by active reabsorption of the chloride and sodium (Wendt *et al.*, 2007). The sodium concentration in sweat is influenced by sweat rate (Mack & Nadel, 2011). According to Mack *et al.*, it is estimated that the average adult can lose up to 96 mEq of sodium per hour during exercise in the heat (Mack & Nadel, 2011). Typically, athletes performing rigorous exercise in the heat have a sweat rate of approximately 1.0-2.5 L/hour (Wendt *et al.*, 2007). Heat stress presents a major threat to maintenance of body fluid volume and concentration (Mack & Nadel, 2011). Therefore, hydration is a crucial component of reducing heat stress on the human body (Rizzo & Thompson, 2018). Sweat responses are compromised during prolonged exercise in the heat, especially individuals wearing heavy clothing or equipment that inhibits the ability for sweat evaporation (Baker, 2019). During heat exposures, the core

temperature threshold for sweating is decreased as well as the sensitivity/onset of sweating (Periard & Racinais, 2019).

Insert Figure 4 here

Factors that influence physiological responses to heat stress

Along with the various body systems that are involved in thermoregulation, many factors influence the body's ability to dissipate heat. These factors include, but are not limited to, hydration, physical fitness level, age, lack of acclimation, concurrent diseases/conditions, sweat gland dysfunction, medicine, clothing, and previous heat illnesses (Epstein, 1990; Wendt *et al.*, 2007; Hosokawa *et al.*, 2014; Casa *et al.*, 2015; Casadio *et al.*, 2017). Due to individual differences in heat tolerance and predisposing factors, those who exert themselves in the heat for a prolonged period of time may be at an increased risk of experiencing a heat illness (Epstein, 1990). A thorough list of the intrinsic and extrinsic factors can be found in Table 1.

Insert Table 1 here

Heat Illnesses

Exertional heat illnesses (EHI) typically occur during exercise in hot and humid environments, in which the individual is unable to adequately thermoregulate. There are many types of EHI's such as, exercise-associated muscle cramps, heat syncope, heat exhaustion, and exertional heat stroke (Casa *et al.*, 2015). Exertional heat stroke (EHS) is the most severe and one of the leading causes of sudden death in sport (Casa *et al.*, 2015). EHS is categorized as an internal body temperature at or above 40.5 degrees Celsius (Hosokawa *et al.*, 2014; Casa *et al.*, 2015). When heat storage exceeds heat dissipation, it results in EHS. One of the first signs of EHS is loss of central

nervous system function, such as altered consciousness, seizures, and confusion (Casa *et al.*, 2015). The risk of morbidity increases the longer a person's core temperature exceeds 40.5 degrees Celsius (Casa *et al.*, 2015). EHS is a medical emergency as multi-organ system failure is a strong possibility (Casa *et al.*, 2015). Fortunately, exertional heat stroke is 100% survivable by providing appropriate medical coverage at all sporting events for accurate recognition and treatment of the condition (Casa *et al.*, 2015). Heat acclimation/acclimatization can be used as a strategy to prevent EHS from occurring (Casa *et al.*, 2015). The level of HA attained determines the level of exercise intensity that an individual can sustain during exercise in the heat (Armstrong & Maresh, 1991).

Heat Acclimation

What is heat acclimation?

Heat acclimation (HA) refers to repeated periods of heat exposure in an artificial (laboratory) setting (Armstrong & Stoppani, 2002). Heat acclimatization is repeated heat exposure in a natural (outside) environment (Armstrong & Stoppani, 2002). HA is a strategy commonly utilized by athletes in team and individual sports who wish to optimize performance in the heat (Benjamin *et al.*, 2019). HA can also be utilized by laborers and warfighters who are consistently exposed to hot and/or humid environments (Benjamin *et al.*, 2019). Heat acclimation protocols vary heavily by design, however HA typically involves an environmental chamber being set to 35-40 degrees Celsius, 10-90% relative humidity, and up to 90 minutes' exercise duration for anywhere between 8-22 consecutive days (Garrett *et al.*, 2011; Stanley *et al.*, 2015; Pryor *et al.*, 2019a). HA typically requires 8-14 consecutive days of exercise-heat exposure (Armstrong & Stoppani, 2002). Heat acclimation periods can be categorized into three groups, short-term (<7

days), medium-term (8-14), and long-term (>15 days) (Garrett *et al.*, 2011). Literature suggests that protocols lasting greater than 8 days are more effective in attaining heat acclimation than short-term acclimation of <7 days (Tyler *et al.*, 2016).

There are five general induction methods for heat acclimation (Garrett *et al.*, 2011; Tyler *et al.*, 2016; Daanen *et al.*, 2018; Pryor *et al.*, 2019a). These include “controlled hyperthermia” or “isothermal”, “controlled work-rate”, “self-regulated”, “controlled heart rate” or “relative intensity”, and “passive heating” (Garrett *et al.*, 2011; Tyler *et al.*, 2016; Daanen *et al.*, 2018; Pryor *et al.*, 2019a). Examples of the various induction methods can be found in Figure 5 (Daanen *et al.*, 2018). It is common for researchers to only use one induction method during a HA protocol, however it may be beneficial to use multiple methods to obtain the greatest HA adaptations (Pryor *et al.*, 2019a). The most common induction method is constant work rate, according to Garrett *et al.*, however little consensus exists on which induction method is most optimal (Garrett *et al.*, 2011; Tyler *et al.*, 2016). Induction methods are categorized as either active or passive. Passive acclimation involves the use of an external heat source (i.e. sauna, chambers, or hot water) to passively increase the temperature of body tissues at rest (Taylor, 2000). Active acclimation occurs when HA is induced during exercise in a hot environment. Limitations exist with each HA induction, see Table 2. One limitation that is true with all induction methods is that maximal potential thermal load may not be achieved. A description of the various induction methods can be found in Table 2.

Insert Figure 5 here

Insert Table 2 here

Heat acclimation causes a complex series of central and peripheral physiological adaptations as well as perceptual changes (Armstrong & Stoppani, 2002; Tyler *et al.*, 2016). The purpose of these adaptations is to reduce the strain on the body that is created during exercise in hot environments and improve cardiovascular stability and thermal tolerance (Casa *et al.*, 2015; Periard *et al.*, 2016; Pryor *et al.*, 2019a). This results in exercise performance enhancements and a reduced risk of heat illnesses (Casa *et al.*, 2015; Periard *et al.*, 2016; Pryor *et al.*, 2019a).

Physiological adaptations

Physiological adaptation is a term used to describe an adjustment in response to a repeated environmental stimulus that enhances the ability of an organism to maintain homeostasis (Armstrong & Stoppani, 2002). The most common physiological adaptation recorded is a reduction in heart rate (HR) (Armstrong & Stoppani, 2002; Periard *et al.*, 2016). Changes in HR are also one of the first adaptations to occur during HR; this is demonstrated in Figure 6 (Periard & Racinais, 2019). In addition to changes in HR, a decrease in the temperature at which sweat production occurs is also observed during HA (Armstrong & Stoppani, 2002). Plasma volume expansion is another common adaptation of HA, typically occurring within the first 3-4 days of heat exposure (Armstrong & Maresh, 1991; Patterson *et al.*, 2004; Garrett *et al.*, 2011; Periard *et al.*, 2016). Approximately two-thirds of improvements resulting from plasma volume expansion are seen over the course of 4-6 days of HA (Pandolf, 1998). Plasma volume expansion has two major advantages (Periard *et al.*, 2016). One advantage is that it increases vascular filling pressure to provide cardiovascular stability (Periard *et al.*, 2016). Another advantage is that it increases the temperature of blood, which aids in heat transfer from the core to the skin (Periard *et al.*, 2016). Plasma volume expansion may not be a marker of heat acclimation in itself,

however in conjunction with hematological adaptations, it proves to be a very valuable multifactorial enhancement of heat acclimation (Racinais *et al.*, 2014).

Insert Table 3 here

Table 3 demonstrates common physiological adaptations seen during and after a period of heat acclimation including increased blood flow, decreased sweat sodium concentrations, and decreased core body temperature (Armstrong & Maresh, 1991; Pandolf, 1998; Armstrong & Stoppani, 2002; Donaldson *et al.*, 2003; Wendt *et al.*, 2007; Mack & Nadel, 2011; Periard *et al.*, 2016; Tyler *et al.*, 2016). A majority of these physiological adaptations develop within the first 4 to 7 days of HA (Periard *et al.*, 2016). The clinical signs that result from HA are all related to autonomic nervous control (Armstrong & Stoppani, 2002). A list of the various perceptual, physiological, and performance adaptations can be found in Figure 7 (Pryor *et al.*, 2019a).

Insert Figure 6 here

Sweat Responses

Sweat responses or sudomotor function, are improved during heat acclimation (Cotter *et al.*, 1997). This includes the threshold for the onset of sweating and sweat concentrations. According to Armstrong and Maresh, changes in sweat response occur within at least 7 days of HA (Armstrong & Maresh, 1991). Sweat response is one of the last adaptations to occur (Periard & Racinais, 2019). This is represented in Figure 6 (Periard & Racinais, 2019). Heat acclimation induces a reduced sweat threshold, meaning that the onset of sweating begins earlier during exercise (Cotter *et al.*, 1997). This means that the body is able to dissipate heat more quickly in a heat acclimatized individual versus an unacclimatized individual. More recent literature

demonstrates that heat acclimation improves local cutaneous vascular responses (Periard *et al.*, 2015).

Heart Rate

It is well documented that heat acclimation reduces heart rate (HR) (Moran *et al.*, 1996; Periard *et al.*, 2016; Rizzo & Thompson, 2018). As stated earlier, cardiovascular strain is reduced during HA due to heart rate adaptations (Garrett *et al.*, 2011). Changes in heart rate are also commonly attributed to plasma volume expansion (Garrett *et al.*, 2011). Initially, exercise in temperate environments prior to HA causes an increase in HR and decrease in stroke volume (Sawka *et al.*, 2011). However, following HA, stroke volume is increased due to the cardiovascular adjustments made to reduce cardiovascular strain (Sawka *et al.*, 2011). This in turn decreases HR as the heart is able to fill more efficiently (Sawka *et al.*, 2011). It is suggested that HR increases to offset the decrease in stroke volume while maintaining cardiac output (Rizzo & Thompson, 2018). Heart rate responses to HA occur relatively quickly, within the first 4 to 5 days (Sawka *et al.*, 2011). The magnitude of HR changes depends largely on exercise intensity, exercise duration, and heat stress (Periard *et al.*, 2016). Furthermore, research has determined that dehydration has a negative effect on HR and core temperature during exercise in the heat (González-Alonso *et al.*, 1995; Sawka *et al.*, 2011). Reduced HR not only coincides with plasma volume expansion, greater stroke volume, and increased cardiac output, but also increases myocardial efficiency (Lorenzo *et al.*, 2010). A decrease in heart rate allows for the heart to not fatigue as quickly as it otherwise would have during heat exposure to the heat (Rizzo & Thompson, 2018). Due to such changes, decreased HR greatly enhances exercise performance (Lorenzo *et al.*, 2010).

Insert Figure 7 here

Perceptual Adaptations

Along with physiological responses that occur as a result from HA, there are also perceptual responses. Thermal sensation is a common variable that is measured during heat acclimation studies, in which the participant reports how hot or cold they feel before, during, and after exercise in the heat. Significant decreases in reported thermal sensation have been reported in numerous studies following a HA protocol (Racinais *et al.*, 2014). A reduction in thermal sensation is important for those exercising in the heat as it adds a level of comfort for the individual. The magnitude of both physiological and perceptual adaptations is largely dependent on environmental conditions, exercise duration, exercise frequency, and amount of exposure to heat (Periard *et al.*, 2016).

Heat acclimation decay

Unfortunately, physiological adaptations gradually dissipate once an athlete is removed from repeated heat exposures (Wendt *et al.*, 2007). According to Armstrong and Maresh, the first adaptations to occur during HA are also the first to disappear when HA is not maintained (Armstrong & Maresh, 1991). For example, plasma expansion is one of the earliest adaptations during HA, and it's also one of the first to return to baseline when removed from heat exposures (Garrett *et al.*, 2011).

There is much variation in current literature regarding the rate at which the physiological adaptations from HA are lost or decay. Many studies show that HA is better retained using dry-

heat acclimation rather than humid-heat acclimation (Pandolf, 1998). There are conflicting findings suggesting that one day of exercise in the heat is required for every five days without heat exposure, while others suggest that one day is lost for every two days spent not in the heat (Periard *et al.*, 2016). For example, one study done by Pryor *et al.*, showed that when the intermittent exercise-heat exposure (IHE) group was compared to the control group, the decay of the physiological adaptations was significantly reduced (Pryor *et al.*, 2019b). This is demonstrated in Figure 8 (Pryor *et al.*, 2019b). Due to the inconsistent data on rate of decay, the typical adaptations to heat have been shown to return to baseline within 3 weeks following cessation of HA (Garrett *et al.*, 2011).

In general, it is believed that the longer that the period of heat acclimation is, the longer the physiological adaptation will remain. However, a study written by Pandolf *et al.* found the rate of decay to be very slow following 18 days after being removed from the heat (Pandolf *et al.*, 1977; Armstrong & Maresh, 1991). These findings were likely attributed to the high cardiorespiratory fitness (measured by VO_{2max}) of subjects had prior to experimentation (Pandolf *et al.*, 1977). This may suggest that higher levels of aerobic fitness are associated with greater heat acclimation retention (Pandolf, 1998).

Insert Figure 8 here

The rate of decay of HA adaptations may depend on factors such as fitness level, environmental conditions, and intensity/duration of training following HA (Lorenzo *et al.*, 2010; Garrett *et al.*, 2011). Other influential factors may include the number of heat exposures per week, the degree

at which core body temperature is increased, and individual's pre-existing natural HA (Pandolf *et al.*, 1977; Armstrong & Maresh, 1991).

Performance Enhancements

It is prevalent in the current literature that heat acclimation induces exercise performance benefits. "Performance" can be defined as the result of established tests that measures exercise ability (Benjamin *et al.*, 2019). Performance benefits include increased time to exhaustion, faster race time, or improved aerobic capacity (Benjamin *et al.*, 2019). Exercise performance enhancements are achieved through the expansion and increase of plasma volume, enhanced sweating, increased blood flow, and improved thermal tolerance (Periard *et al.*, 2016). An increase in plasma volume response induced by heat acclimation causes an indirect increase in cardiac output, resulting in improved sport performance (Racinais *et al.*, 2014). A recent meta-analysis discovered that the greatest performance improvement following HA was time to exhaustion, followed by improvement in time trials (Benjamin *et al.*, 2019). This is demonstrated in Figure 9 (Benjamin *et al.*, 2019). Another meta-analysis found that exercise capacity was improved on average 23% following HA (Tyler *et al.*, 2016). One of the most common tests used to measure exercise capacity is maximal oxygen uptake or $\text{VO}_{2\text{max}}$. $\text{VO}_{2\text{max}}$ tests are commonly used because they are easy to administer, economic, and fairly accurate when performed correctly.

Aerobic Performance Enhancements

Maximal Oxygen Uptake (VO_{2Max})

One of the major improvements that occurs during heat acclimation is increased aerobic fitness, measured by VO_{2max} testing (Garrett *et al.*, 2011; Sawka *et al.*, 2011). VO_{2max} represents the amount of oxygen that the body utilizes while at maximum physical effort (Shete *et al.*, 2014). This is measured by analyzing the inspired and expired air during exercise (Shete *et al.*, 2014). Typically, individuals with a higher VO_{2max} , have a greater aerobic capacity and ability to perform better at endurance events. The average VO_2 max for males is anywhere from 27 to 51 $ml \cdot kg^{-1} \cdot min^{-1}$ (Astrand, 1960). A detailed list of average VO_{2max} ranges for males can be found in Table 4 (Astrand, 1960). Originally, acute heat stress reduces VO_{2max} measurements when compared to those performed in a temperate environment (Sawka *et al.*, 1985). However, once heat acclimation is induced, VO_{2max} significantly improves (Sawka *et al.*, 1985). One study found that VO_{2max} increased approximately 8% following a 10-day HA protocol (Lorenzo *et al.*, 2010).

Insert Table 4 here

It is well established that cardiorespiratory fitness level along with an individual's predisposition to suffering from heat illness, plays a substantial role in the body's ability to thermoregulate (Pandolf *et al.*, 1977; Benjamin *et al.*, 2019). The rate at which heat acclimation occurs is directly related to one's level of cardiorespiratory fitness (Armstrong & Maresh, 1991). Increases in VO_{2max} during HA may be due to improved fitness that is induced by exercise training alone (Brooks, 1984).

In an early study by Sawka *et al.*, it was suggested that the initial reduction in $\text{VO}_{2\text{ max}}$ is due to the participants having a greater ability to increase cardiac output during maximal exercise in the heat (Sawka *et al.*, 1985). This was further proved when Pandolf *et al.* demonstrated improvements in aerobic exercise are associated with elevations in core temperature and sweat rate, which are mechanisms to improve thermoregulatory responses during heat stress (Pandolf, 1998). The relationship between aerobic capacity or oxygen consumption is directly proportional to cardiac output (Periard *et al.*, 2016). The effects of HA on cardiac output and aerobic capacity are dependent on exercise intensity and the severity of heat stress (Periard *et al.*, 2016). Therefore, $\text{VO}_{2\text{ max}}$ results can vary in magnitude based on the heat acclimation protocol and hot/humid conditions and the duration of the exercise. Further, it is proved that increases in performance are relative to increases in $\text{VO}_{2\text{ max}}$ (Benjamin *et al.*, 2019).

Gaps in the Literature

To our knowledge, no study to date has assessed aerobic performance in dual heat acclimatized individuals, such as a protocol that utilizes both natural and laboratory settings to induce physiological and perceptual adaptations. Furthermore, many studies use participants who have a “low” or “untrained” fitness level, there has not been much literature that includes highly trained individuals (Benjamin *et al.*, 2019). In addition, we set out to explore potential reasoning behind why some studies recorded significant improvement in $\text{VO}_{2\text{ max}}$ and others did not (Benjamin *et al.*, 2019). Lastly, it’s been well documented in the literature that HA protocols are often inconsistent among studies (Benjamin *et al.*, 2019). Factors like induction length, induction method, exercise duration and intensity, and heat index during induction all vary between studies

(Benjamin *et al.*, 2019). However, we aimed to utilize various induction methods to help mitigate these gaps in the literature.

Conclusion

Thermoregulation during heat stress is a complex system that requires many body systems to work together to protect the body from thermal strain and heat illness. The CNS and cardiovascular systems endure the majority of thermal strain during times of acute heat stress. However, when an individual undergoes a heat acclimation protocol the body is able to adapt physiologically and perceptually to enhance performance and thermal comfort.

Chapter Two: Introduction

It has been well documented in the literature that heat acclimation (HA) increases maximal aerobic power ($\text{VO}_{2\text{max}}$), thus improving exercise performance (Nadel *et al.*, 1974; Sawka *et al.*, 1985; Lorenzo *et al.*, 2010). Numerous physiological adaptations have been linked to the improvement in aerobic power during HA including reduced oxygen uptake at a given power output, reduced blood lactate at a given power output, and plasma volume expansion (Young *et al.*, 1985; Lorenzo *et al.*, 2010). In an early study done by Pivarnik *et al.*, an increase in red blood cell volume was seen following heat acclimation, likely contributing to enhanced oxygen carrying capacity and increased peak $\text{VO}_{2\text{max}}$ results (Pivarnik *et al.*, 1987). Recent theories suggest that increased changes in $\text{VO}_{2\text{max}}$ during HA protocols are in part due to the exercise training portion alone, rather than repeated heat exposure (Brooks, 1984).

Aside from aerobic performance enhancements during heat acclimation, little is known about how long performance remains increased. Once an individual has been removed from repeated heat exposures, the adaptations including performance enhancement, gradually dissipate (Wendt *et al.*, 2007). Sustaining cardiovascular and thermoregulatory adaptations that result from HA is vital to the safety of individual's who physically exert themselves in hot environments on a regular basis (Pryor *et al.*, 2019b). Due to differences among individual study designs, a set rate of decay has not yet been established. Individual fitness level and aerobic capacity may also influence rate of decay (Lorenzo *et al.*, 2010). A study by Pandolf *et al.*, revealed no significant decreases in $\text{VO}_{2\text{max}}$ measurements following 2 ½ weeks without heat exposure (Pandolf *et al.*, 1977). The authors hypothesized that the lack of change was likely from the participant's high aerobic capacity prior to the experiment (Pandolf *et al.*, 1977).

Much of the literature differs regarding the length of time in which adaptations and enhancements remain following heat acclimation. To date, no studies have determined the rate at which aerobic performance decays, if at all, once removed from repeated heat exposures. The aim of this study is to determine the impact that heat acclimation has on maximal aerobic capacity in endurance runners and identify how long these changes remained once heat exposures are removed. We hypothesized that $\text{VO}_{2\text{max}}$ will not increase following heat acclimatization or heat acclimation and will remain unchanged after removal of heat exposures.

Chapter Three: Methodology

Participants

Twenty-seven male endurance runners participated in this study (age: 36 ± 12 years, nude body mass: 73.03 ± 8.97 kg, height: 178.81 ± 6.39 cm, VO_{2max} : 57.48 ± 7.03 ml·kg⁻¹·min⁻¹). The selection criteria included male endurance athletes between ages 18 and 55. The protocol was approved by the University of Connecticut Institutional Review Board and all participants provided written informed consent prior to participation. Each participant completed 5 VO_{2max} tests, 5 treadmill running exercise tests, a summer training regimen, and 5 days of heat acclimation. Participants were randomly assigned to one of three groups for the heat acclimation maintenance portion of the study following test 3, once a week (n=9), twice a week (n=10), or not at all/control (n=8).

Testing Trials

All testing trials were performed in a heat chamber laboratory setting (ambient temperature [T_{amb}], $35.42 \pm 1.06^\circ\text{C}$; relative humidity [%RH], $46.35 \pm 2.48\%$; Wet Bulb Globe Temperature [WBGT], 29.62 ± 1.37 ; wind speed, 3.98 ± 0.30 mph). Trials consisted of 60 minutes of steady state exercise ($59.12 \pm 1.74\%$ VO_{2max}) on a motorized treadmill (T150; COSMED, Traunstein, Germany), at 5 points throughout the study: baseline (Test 1), prior to HA (Test 2), immediately following HA (Test 3), during heat acclimation maintenance (Test 4), and post- heat acclimation maintenance (Test 5). Tests 4 and 5 varied depending on which experimental group the participant was assigned, however both tests were during or after the maintenance period. Heart rate (HR), rectal temperature (T_{rec}), rate of perceived exertion (RPE), thermal sensation (TS), thirst, and fatigue were recorded every five minutes throughout testing trails. HR was measured with a polar chest strap (H10®, Polar Electro™, Kempele, Finland) and rectal temperature was recorded by having participant's self-insert a rectal probe 10-15cm passed the anal sphincter

(MP160; BIOPAC Systems Inc., Goleta, CA, USA). Skin temperature (T_{sk}) was measured at the thigh, calf, chest, and upper arm (iButton; iButton Link LLC., Whitewater, WI, USA). Sweat rate (SR) was calculated using nude body mass measurements pre and post exercise. Proper hydration prior to the start of testing was ensured with the measuring of urine specific gravity (USG) and urine color chart. Participants were restricted from drinking fluid during the 60-minute exercise session.

Following the 60 minutes of exercise, participants were cooled to 38.5°C and instructed to drink water to replace fluids lost. Nude body mass was measured post fluid consumption to confirm sufficient fluid replacement by return to baseline nude body mass. Participants were then asked to complete a 4k time trial at their own pace. HR and T_{rec} were measured at every kilometer during the 4k time trial.

Field Testing/Heat Acclimatization

Following baseline testing, a summer (June-August) training program was initiated. We can safely assume that the subjects were not heat acclimatized at this time due to the climate in New England prior to the summer months. During the summer training period, all participants were responsible for recording training data, such as HR, distance and location, using their own wearable training devices and respective tracking software (Garmin, [Forerunner® Fenix® Vivoactive® Garmin™ Ltd., Olathe, Kansas, USA; Polar H10 and Polar Beat application, [H10®, Polar Electro™, Kempele, Finland]]).

Heat Acclimation

Following test 2, participants completed a 5-day heat acclimation (HA) protocol in the heat chamber (ambient temperature [T_{amb}], $39.13 \pm 1.37^{\circ}\text{C}$; relative humidity [%RH], 51.04 ± 8.42 %; Wet Bulb Globe Temperature [WBGT], 33.16 ± 1.95 ; wind speed, 00 mph). In attempt to keep the days of HA continuous, all 5 sessions were completed within 8 days. The days between each test are presented in Table 5. Each heat acclimation session induced maximal hyperthermia for 60 minutes. Maximal hyperthermia was defined as rectal temperature between 38.50°C and 39.75°C . Exercise began at 70% of the individual's $\text{VO}_{2\text{max}}$, and intensity was adjusted throughout the session to keep the participants within the maximal hyperthermia range.

Insert Table 5 here

Heat Acclimation Maintenance (HAM)

Following the 5-day HA protocol, participants were required to complete a HAM session as often as once a week (1x), twice a week (2x), or not at all (con), depending on which group they were assigned to. The exercise session was the same as one heat acclimation session, consisting of 60 minutes at maximal hyperthermia. Environmental conditions remained the same as the HA protocol conditions.

$\text{VO}_{2\text{max}}$ Tests

$\text{VO}_{2\text{max}}$ tests were performed at the following time points for both experimental groups (baseline, test 1; post-HAz, test 2; post-HA, test 3; 4 weeks of HAM, test 4; 8 weeks of HAM, test 5). The

control group did not perform any HAM sessions. A timeline of the $\text{VO}_{2\text{max}}$ testing can be seen in Figure 10.

Insert Figure 10 here

Each metabolic cart was calibrated before participants arrived to ensure measurement accuracy. Each participant was fitted for a mask (Hans Rudolph Inc., Shawnee, KS), and the mask was assembled with a breathing tube that attached to the metabolic cart (Hans Rudolph, Inc., Shawnee, KS). The $\text{VO}_{2\text{max}}$ protocol consisted of 2-minute stages, in which the speed was increased 0.5-1.0 mph based on the participants' comfort level. Participants were given a 5-minute warm up prior to beginning. During each test, $\text{VO}_{2\text{max}}$, velocity at $\text{VO}_{2\text{max}}$ (vVO_2), maximum heart rate (HR), Rate of Perceived Exertion (RPE), and Respiratory Exchange Ratio (RER) were measured. All variables were recorded at each 2-minute stage of the test. $\text{VO}_{2\text{max}}$ and RER were calculated automatically using the ParvoMedics system (TrueOne, Salt Lake City, Utah). The test was completed once the participant wished to terminate due to fatigue or discomfort.

Data Analysis

Statistical analysis was completed using SPSS Statistics version 25 (IBM Corp. Armonk N.Y., USA). Differences in $\text{VO}_{2\text{max}}$, vVO_2 , and max HR for tests 1-3 were analyzed using one-way ANOVAs. Differences in $\text{VO}_{2\text{max}}$, vVO_2 , and max HR between different time points and groups during tests 3-test 5 were analyzed using repeated measure ANOVAs. If Mauchly's Test for Sphericity was deemed significant (sphericity assumed), Greenhouse-Geisser was used in

correction. Cohen's d effect size was used for tests 1-3. Hedge's g was used for tests 3-5 because sample sizes varied. Pairwise differences were analyzed post-hoc with Bonferroni corrections, to reduce the risk of type 1 errors. All measurements are expressed as means with standard deviations (mean \pm SD), mean differences (MD), 95% confidence intervals (CI) and effect size (ES). ES was described using the following: <0.2 = insignificant, $0.2-0.6$ = small, $0.7-1.1$ = moderate, $1.2-2.0$ = large, and >2.0 = very large. Statistical significance was defined as $p<0.05$, a priori.

Of the twenty-seven participants, twenty-four participants were included in analysis of the VO_{2max} data due to equipment malfunctions and/or inability for the subject to perform the test (i.e. illness). The HAM groups subject count was as follows: 1x ($n=8$), 2x ($n=9$), and con ($n=7$). Heart rate was averaged for sub14, test 3. Test 4 was averaged for sub16 and sub22. Test 4 VO_{2max} results were averaged for sub36. Test 5, VO_{2max} results were averaged for subjects 35, 13, 20, and 24. Test 5 was averaged for subject 7. Subject 17 tests 1-3 were completely removed due to equipment malfunction, and tests 3-5 were averaged using all subject's data for power purposes.

Chapter Four: Results

VO_{2max}

VO_{2max} data are presented in Table 6 for Tests 1 through 3. VO_{2max} data for Tests 3 through 5 are presented in Table 7. There were no significant interaction differences between Test 1, Test 2, and Test 3 ($p = 0.363$; Figure 11). Effect sizes between Test 1 vs. Test 2, Test 2 vs. Test 3, and Test 1 vs. Test 3 were small (ES: 0.23, 0.02, and 0.23, respectively). There were no group by time effects for Test 3, Test 4, and Test 5 ($p=0.671$; Figure 12).

Insert Table 6 here

Insert Table 7 here

Insert Figure 11 here

Insert Figure 12 here

vVO_2

Velocity at VO_{2max} data for Tests 1 through 3 are presented in Table 8. Data for Test 3 through 5 are presented in Table 9. There were no significant interaction differences between Test 1, Test 2, and Test 3 ($p = 0.09$; Figure 13). Effect sizes between Test 1 and Test 2, Test 2 and Test 3 and Test 1 and Test 3 were small (ES: 0.18, 0.18, and 0.00, respectively). There were no group by time effects for Test 3, Test 4, and Test 5 ($p=0.215$; Figure 14).

Insert Table 8 here

Insert Table 9 here

Insert Figure 13 here

Insert Figure 14 here

Heart Rate

Heart rate at $\text{VO}_{2\text{max}}$ data for Tests 1 through 3 are presented in Table 10. Heart rate data for Tests 3 through 5 are presented in Table 11. The one-way ANOVA revealed there were significant differences between time points 1-3 ($p=0.006$). There were significant differences between Test 1 and Test 3 (ES: 0.43, $p<0.001$; Figure 15). There were no significant differences between Test 1 and 2 (ES:0.24, $p=0.33$), and Test 2 and Test 3 (ES:0.2, $p=0.57$). There were no group by time effects for Test 3, Test 4, and Test 5 ($p=0.59$; Figure 16).

Insert Table 10 here

Insert Table 11 here

Insert Figure 15 here

Insert Figure 16 here

In another paper with the same participants, the status of heat acclimation was confirmed with recorded improvements in heart rate (HR), rectal temperature (T_{rec}), and sweat rate (SR), $p < 0.05$. We also reported significant improvements in T_{rec} and HR following heat acclimation and heat acclimatization. Whereas, the only improvements in SR followed heat acclimation (CL Benjamin 2020).

Chapter Five: Discussion

In this study, no changes in $\text{VO}_{2\text{max}}$ were found following a heat acclimation protocol. Our observations were that $\text{VO}_{2\text{max}}$ was unchanged following a heat acclimation protocol. These findings are inconsistent with much of the existing literature (Pivarnik *et al.*, 1987; Lorenzo *et al.*, 2010; Keiser *et al.*, 2015; James *et al.*, 2017; Rendell *et al.*, 2017; Willmott *et al.*, 2018). One study reported a 12.4% increase in $\text{VO}_{2\text{max}}$ (Pivarnik *et al.*) while two others reported 8% and 9.6% increases in $\text{VO}_{2\text{max}}$ (Lorenzo *et al.*, 2010) (Keiser *et al.*, 2015) following HA protocols. Additionally, during a recent meta-analysis, 7 out of 12 $\text{VO}_{2\text{max}}$ tests observed performance improvements following HA (Benjamin *et al.*, 2019). Our findings are in accordance with our hypothesis that we would not see a change in $\text{VO}_{2\text{max}}$ after HA and after HAM. Therefore, we can speculate the reasoning behind our results is due to starting fitness levels, running economy, recovery time between tests, and environmental conditions during $\text{VO}_{2\text{max}}$ tests.

Initial Fitness Level

The increases in $\text{VO}_{2\text{max}}$ reported by many other studies may be due to increased fitness levels that were induced by the exercise training alone rather than heat acclimation specifically (Brooks, 1984). This may be demonstrated in Figure 11 and Figure 12 when looking at the twice a week heat training group, as there is a slight increase for each test. These individuals completed two times more exercise than the other groups, potentially explaining the increase found among tests. A study by Pivarnik *et al.* saw the greatest increase in aerobic performance in “untrained” subjects with a 12.4% increase in $\text{VO}_{2\text{max}}$. The average $\text{VO}_{2\text{max}}$ prior to HA for all “untrained” subjects was $44.2 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ (Pivarnik *et al.*, 1987). The dramatic increases in $\text{VO}_{2\text{max}}$ were likely attributable to the significant amount of improvement possible, due to the participants’

lower level of fitness exhibited prior to the exercise training. As seen in Figures 10 and 11, there were no significant differences between any of the $\text{VO}_{2\text{max}}$ tests in any of the groups throughout the duration of our study. This may be due to the fact that our subjects had such a high $\text{VO}_{2\text{max}}$ before the experimental protocol, $57.48 \pm 7.03 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$, and participants were accustomed to high intensity training. It is understandable how an untrained individual would likely see the majority of performance and aerobic improvements following HA, just as Pivarnik et al. demonstrated (Pivarnik *et al.*, 1987).

Running Economy

Another possible reason for the lack of differences in $\text{VO}_{2\text{max}}$ between tests is due to participant's running economy. Running economy (RE) is defined as the relationship between oxygen consumption and velocity of running (Daniels & Daniels, 1992). It is calculated using the steady-state consumption of oxygen and the respiratory exchange ratio (Saunders *et al.*, 2004). Running economy is considered an important factor in endurance running performance (Daniels & Daniels, 1992; Saunders *et al.*, 2004). It can also determine the difference in performance between individuals with similar $\text{VO}_{2\text{max}}$ measurements (Daniels & Daniels, 1992; Saunders *et al.*, 2004). This explains the differences in vVO_2 and HR max found in individuals with similar $\text{VO}_{2\text{max}}$ measurements. Individuals with a good RE use less energy and oxygen than those with a poor RE, while at the same velocity (Saunders *et al.*, 2004). Daniels et al. claim that velocity at $\text{VO}_{2\text{max}}$ (vVO_2) is a better predictor of running success than $\text{VO}_{2\text{max}}$ or RE because it allows us to see how fast an individual can run while at maximal oxygen uptake (Daniels & Daniels, 1992). However, according to Saunders et al., RE is a better predictor of performance than $\text{VO}_{2\text{max}}$ in elite runners with similar $\text{VO}_{2\text{max}}$ measurements (Saunders *et al.*, 2004). As demonstrated in

Figure 14, HR decreased between all tests following HA, with a significant difference ($p < 0.001$) found during test 3. It has been shown that there is a correlation between physiological factors (ie. HR) and changes in RE, and the potential that they are inversely proportional (Saunders *et al.*, 2004). This further explains why we did not see changes in $\text{VO}_{2\text{max}}$ or vVO_2 , but did see changes in HR. A decrease in HR can also be explained simply by demonstrating that HA was induced successfully (King *et al.*, 1985).

Rest between tests

Another possible explanation as to why we did not see changes in $\text{VO}_{2\text{max}}$ could be lack of recovery time between $\text{VO}_{2\text{max}}$ tests and testing trials. Demonstrated in a study by Daanen *et al.*, reduction in core body temperature was not seen until three to seven days following HA (Daanen *et al.*, 2011). This further suggests that optimal performance changes may not occur for up to one week following a period of rest after heat acclimation. During our study, the average amount of days between heat acclimation and $\text{VO}_{2\text{max}}$ testing was 10 days, ranging from 5 to 21 days. Another recent study suggested that 96 hours of rest following a training regimen was optimal for seeing improved performance (Waldron *et al.*, 2019). Based on these findings by Daanen *et al.* and Waldron *et al.*, we can assume that we may have seen improvements in $\text{VO}_{2\text{max}}$, especially following HA, had we provided more consistent rest days between tests and exercise protocols (Daanen *et al.*, 2011; Waldron *et al.*, 2019). Similar to our study, Febbraio *et al.* conducted a HA study with participant's who were highly fit individuals with an average $\text{VO}_{2\text{max}}$ of $68.1 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ (Febbraio *et al.*, 1994). The authors, however, saw decreases in $\text{VO}_{2\text{max}}$ results following HA (Febbraio *et al.*, 1994). During their study $\text{VO}_{2\text{max}}$ tests were performed

less than 24 hours after HA. This further supports the theory that recovery time between tests allows for optimal performance enhancements (Daanen *et al.*, 2011).

Environmental Conditions of Testing

A majority of the literature that found increases in $\text{VO}_{2\text{max}}$ following HA performed the $\text{VO}_{2\text{max}}$ tests in warm or hot environments (Lorenzo *et al.*, 2010; Keiser *et al.*, 2015; Willmott *et al.*, 2018). The participants had been successfully heat acclimated in each study, therefore we can assume that performance in the heat and thermal perception during $\text{VO}_{2\text{max}}$ would have improved (James *et al.*, 2017). In a study by Lorenzo *et al.*, it was shown that $\text{VO}_{2\text{max}}$ increased 8% in a hot environment compared to 5% in a cool environment (Lorenzo 2010). Controversially, Takeno *et al.* demonstrated an increase in $\text{VO}_{2\text{max}}$ measurements following HA, when tested in both a hot and cool environment (Takeno *et al.*, 2001). This further suggests a training effect is responsible for increased $\text{VO}_{2\text{max}}$, rather than heat acclimation alone. One study that found an increased $\text{VO}_{2\text{max}}$ by 4.1%, $\text{VO}_{2\text{max}}$ was performed in a hot environment (Willmott *et al.*, 2018). The authors attributed this increase mainly to hypervolemia and the increased cardiac output following HA (Lorenzo *et al.*, 2010; Willmott *et al.*, 2018). One theory proposed by James *et al.* suggests that during cooler conditions, muscle temperature is decreased which requires a greater oxygen consumption for a given running speed (James *et al.*, 2017). Scientific evidence is needed to support this theory (James *et al.*, 2017).

In our study, we performed all $\text{VO}_{2\text{max}}$ tests in a thermo-neutral environment. We might have seen changes in $\text{VO}_{2\text{max}}$ had we measured them in a hot environment, similar to the three studies that did and saw improvements (Lorenzo *et al.*, 2010; Keiser *et al.*, 2015; Willmott *et al.*, 2018).

In addition, each of these three studies had used a 10-day heat acclimation protocol, whereas we administered a 5-day heat acclimation protocol. This might also explain why we did not see significant improvement in $\text{VO}_{2\text{max}}$ results.

Conclusion

In this study, we found that there were no significant changes in aerobic performance among endurance runners following heat acclimation/heat acclimatization and intermittent exercise heat exposures. Our findings suggest that this lack of improvement in aerobic performance is likely due to the high starting fitness levels of the participants. Other factors that could have contributed to this include enhanced running economy of athletes at the elite level, recovery time between tests, and the environmental conditions at which the $\text{VO}_{2\text{max}}$ tests were performed. Our results were consistent with our hypotheses that $\text{VO}_{2\text{max}}$ would not increase following heat acclimation and would remain unchanged after removal of heat exposures. As demonstrated in Table 12, when the results of this study were compared to another study that measured $\text{VO}_{2\text{max}}$ results following HA, it is clear that further research may provide us with answers to why $\text{VO}_{2\text{max}}$ data is inconsistent (Aoyagi *et al.*, 1998).

Legends

Figure 1. Heat transfer mechanisms. Zhang 2017

Figure 2. Model of Human Thermoregulation. Belval

Figure 3. Threshold Onset Changes before and after heat acclimation. Periard 2015

Figure 4. Thermoregulatory negative feedback loop. Bongers 2020

Figure 5. Heat Acclimatization/acclimation Induction Methods. Daanen et al. 2018

Figure 6. Time course of Adaptations, Periard et al 2019.

Figure 7. Physiological and Perceptual Adaptations during Heat Acclimation. Pryor et al 2019.

Figure 8. Group comparison of adaptation decay after 25 days removed from the heat. Pryor et al. 2019b.

Figure 9. Improvements in Performance following heat acclimation. Benjamin et al. 2019

Figure 10. $\text{VO}_{2\text{max}}$ test timeline.

Figure 11. Individual participant VO_2max measurements for Tests 1-3.

Figure 12. Average VO_2max measurements for Tests 3-5.

Figure 13. Individual participant vVO_2max measurements for Tests 1-3.

Figure 14. Average vVO_2max measurements for Tests 3-5.

Figure 15. Individual participant maximum heart rate measurements for Tests 1-3.

**Significant from Test 1*

Figure 16. Average maximum heart rate measurements for Tests 3-5.

Tables

Table 1. Factors that influence heat illness

Intrinsic Factors	Extrinsic Factors
Fitness level	Clothing (i.e. equipment, uniforms, etc.)
Hydration status	Environmental conditions (High ambient temperature, high humidity, WBGT, solar radiation, wind speed)
Sleep deprivation	Inappropriate work-to-rest ratios
Reluctance to report issues	Pre-existing medical conditions
Genetics	Medications/Supplements
Heat acclimation/acclimatization	Exercise Intensity

Adapted from

Table 2. Induction Methods

Induction Method	Description	Limitation(s)
Isothermal/Controlled hyperthermia	Gradually reach a core temperature of 38.5°C, generally. Intensity is adjusted to meet the temperature threshold. Maintain temperature threshold for duration of exercise.	Some people can tolerate higher temperatures, thermal tolerance is on an individual basis. 38.5°C is arbitrarily chosen, more research should be done to show that it is a solid threshold, while greater temperatures could potentially induce more adaptations.
Controlled work-rate	A metric (ex. $\text{VO}_{2\text{ max}}$) is selected to exercise at for the duration of the protocol or until temperature limit is met	If the individual reaches the temperature threshold, the work rate cannot be altered to keep the person exercising for longer.
Self-regulated	Exercise intensity is chosen by the participant based on their thermal comfort	Individual may not get hot enough to induce physiological adaptations
Controlled heart rate (relative intensity)	Level of cardiovascular strain is maintained measured by HR that corresponds with a predetermined relative exercise intensity.	Core temperature could potentially get too high

Passive heating	Occurs while the individual is resting. Typically using hot water immersion or sauna bathing.	Limited research to prove the magnitude of adaptations.
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Adapted from (Tyler *et al.*, 2016; Daanen *et al.*, 2018)

Table 3. Physiological Adaptations to Exercise in the Heat

Parameter	Result
Heart rate	Decrease
Core temperature	Decrease
Plasma volume	Increase
Sweat rate & sensitivity	Increase
Sweat sodium concentration	Decrease
Oxygen consumption	Decrease
Skin temperature	Decrease
Skin blood flow	Increase
Stroke volume	Increase

Adapted from (Sawka *et al.*, 2011; Pryor *et al.*, 2019a)

Table 4. Average male VO₂ max ranges

Age	VO₂ Max (ml·kg⁻¹·min⁻¹)
20-29	44-51
30-39	40-47
40-49	36-43
50-59	32-39
60-69	27-35

Adapted from (Astrand, 1960)

Table 5. Average Days between HA

Test	Days (means±SD)
Test 2-HA1	4±2
HA1- HA2	1±1
HA2-HA3	2±1
HA3-HA4	2±1
HA4-HA5	1±1
Total number of days to complete HA	6 ±1

Table 6. VO₂max average, standard deviation, and total number of subjects (n) for Tests 1-3.

VO ₂ max Test	Mean (ml/kg/min)	SD	n=
1	57.92	6.82	24
2	59.65	8.24	24
3	59.49	7.18	24

Table 7. VO₂max average, standard deviation, total number of subjects (n) for Tests 3-5.

VO ₂ max Test	Group	Mean (ml/kg/min)	SD	n=
3	Con	58.70	8.48	8
	1x	60.96	10.31	9
	2x	57.62	6.59	10
4	Con	59.25	6.70	8
	1x	61.00	11.25	9
	2x	59.55	4.73	10
5	Con	58.28	7.05	8
	1x	59.03	12.66	9
	2x	60.18	6.38	10

Table 8. Velocity at VO₂max average, standard deviation, and total number of subjects (n) for Tests 1-3.

VO ₂ max Test	Mean (mph)	SD	n=
1	10.17	1.00	24
2	9.98	1.06	24
3	10.17	1.03	24

Table 9. Velocity at VO₂max average, standard deviation, and total number of subjects (n) for Tests 3-5.

VO ₂ max Test	Group	Mean (mph)	SD	n=
3	Con	10.34	1.16	8
	1x	9.94	1.29	9
	2x	9.95	0.93	10
4	Con	10.24	1.08	8
	1x	9.89	1.17	9
	2x	10.0	0.63	10
5	Con	9.88	1.13	8

	1x	9.82	1.41	9
	2x	10.1	0.99	10

Table 10. Maximum heart rate at VO₂max average, standard deviation, and total number of subjects (n) for Tests 1-3.

VO ₂ max Test	Mean (bpm)	SD	n=
1	179.71	11.17	24
2	177.17	10.23	24
3	175.17	10.12	24

Table 11. Maximum heart rate at VO₂max average, standard deviation, and total number of subjects (n) for Tests 3-5.

VO ₂ max Test	Group	Mean (bpm)	SD	n=
3	Con	178.63	9.86	8
	1x	176.94	12.82	9
	2x	172.5	10.96	10
4	Con	179.38	11.2	8

5	1x	175.22	11.21	9
	2x	175.5	15.83	10
	Con	177.88	13.21	8
	1x	172.92	13.86	9
	2x	169.7	18.06	10

Table 12. Comparative VO_{2max} Results

Reference	Induction length (days)	Exercise Intensity*	Heat Index HA Induction (°C)	Heat Index VO_{2 max} Testing (°C)	Baseline VO_{2 max} (ml·kg⁻¹·min⁻¹)	Post HA VO_{2 max} (ml·kg⁻¹·min⁻¹)	Δ VO_{2 max} (%)
This Study	5	Moderate/High	53	24	57.9	59.5	+2.76
Aoyagi et al. (1998) A [#]	6	Low	43	43	39.9	46.0	+15.29
Aoyagi et al. (1998) B [^]	6	Low	43	43	43.9	45.7	+4.1
Aoyagi et al. (1998) C [~]	6	Low	43	43	49.5	48.1	-2.83

*Exercise Intensity was defined as either “low”, <55% VO_{2max}, “moderate”, 55-70% VO_{2max}, and “high”, >70% VO_{2max}.

#participants completed 8 week-control no exercise prior to HA

[^]participants completed 8 week-endurance training program prior to HA

[~]during HA, participants wore normal clothing, intermittently while wearing protective clothing

Figures

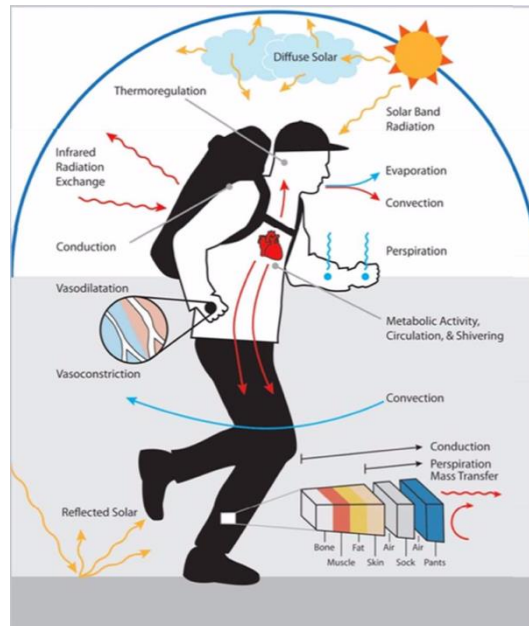


Figure 1. Heat transfer mechanisms (Zhang & Wang, n.d.).

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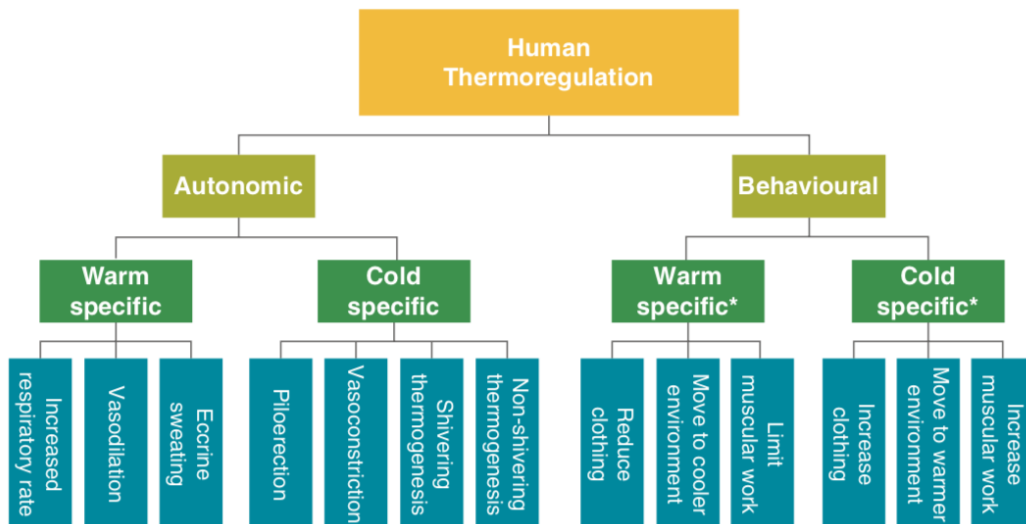


Figure
Model

Human Thermoregulation (Belval, n.d.)

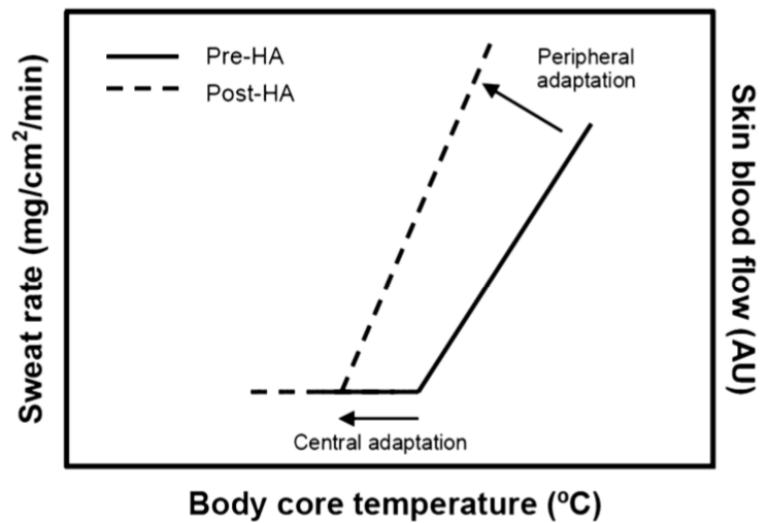


Figure 3. Threshold Onset Changes before and after heat acclimation (Periard *et al.*, 2015).

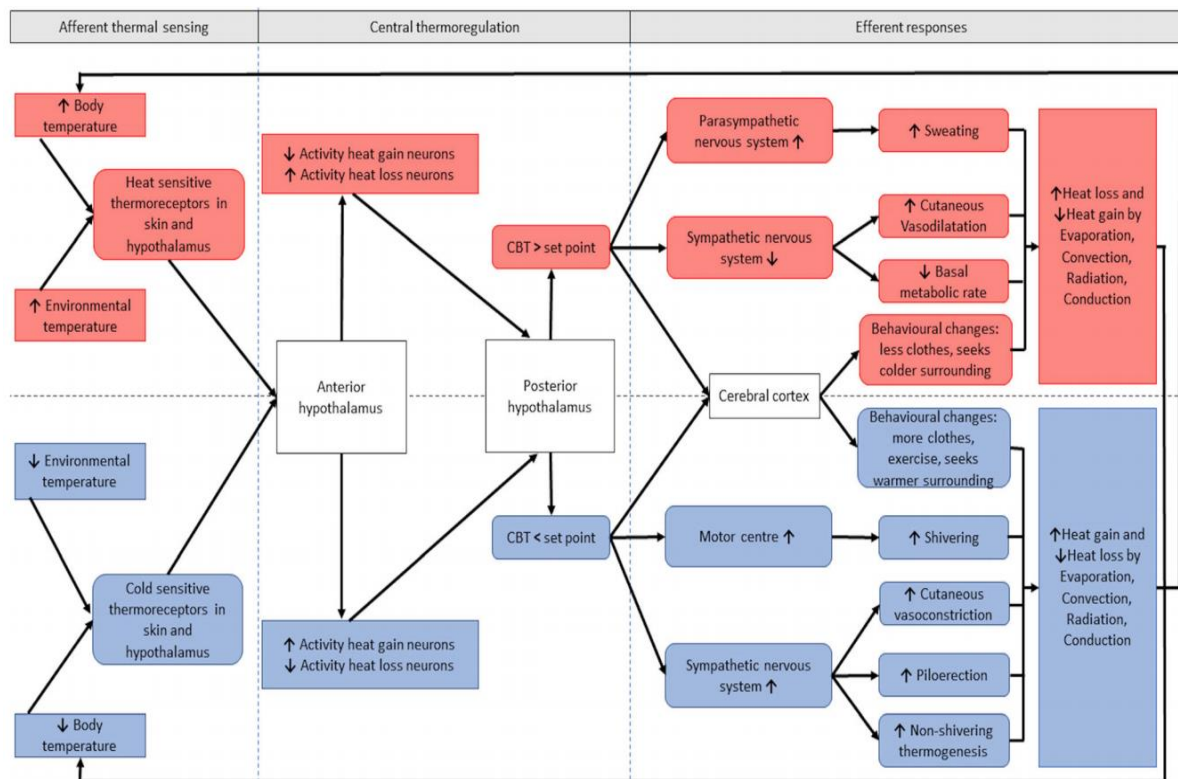


Figure 4. Thermoregulatory negative feedback loop (Bongers *et al.*, 2020).

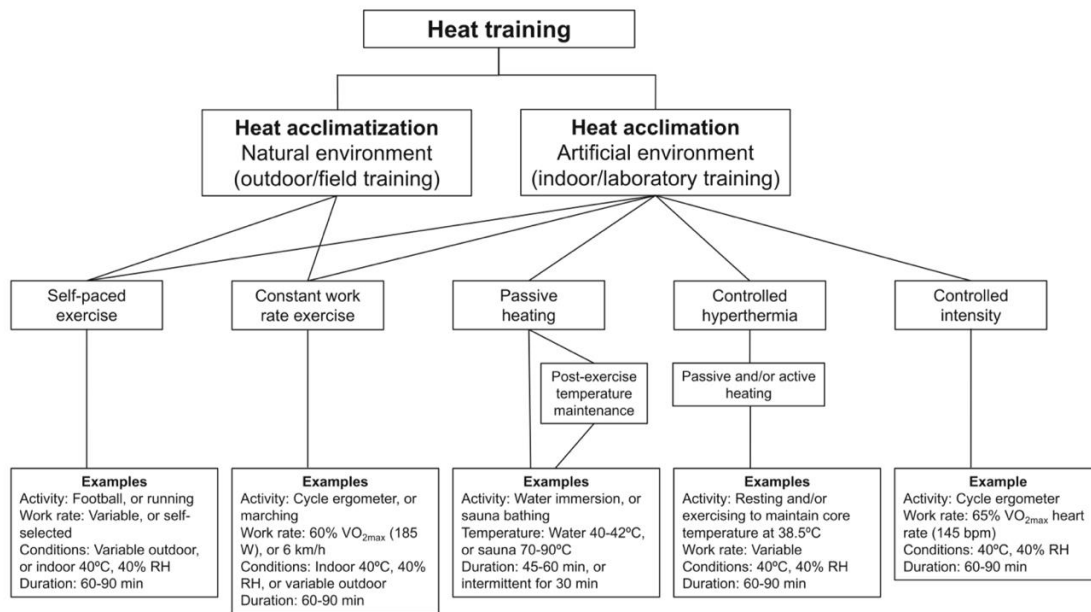


Figure 5. Heat Acclimatization/acclimation Induction Methods (Daanen *et al.*, 2018).

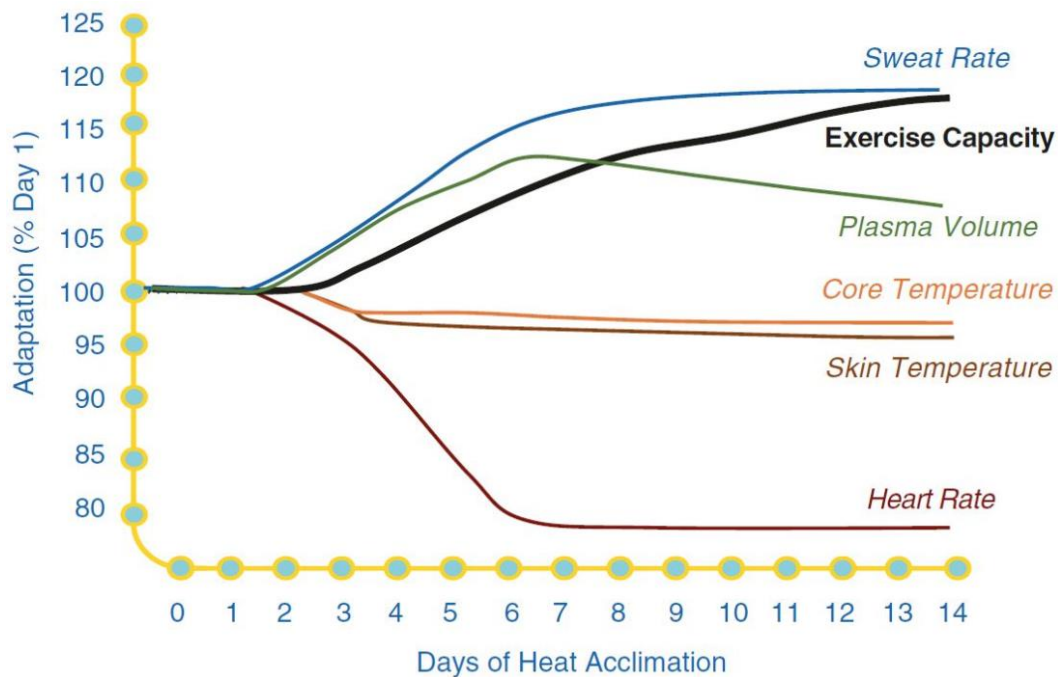


Figure 6. Time course of Adaptations (Periard & Racinais, 2019).

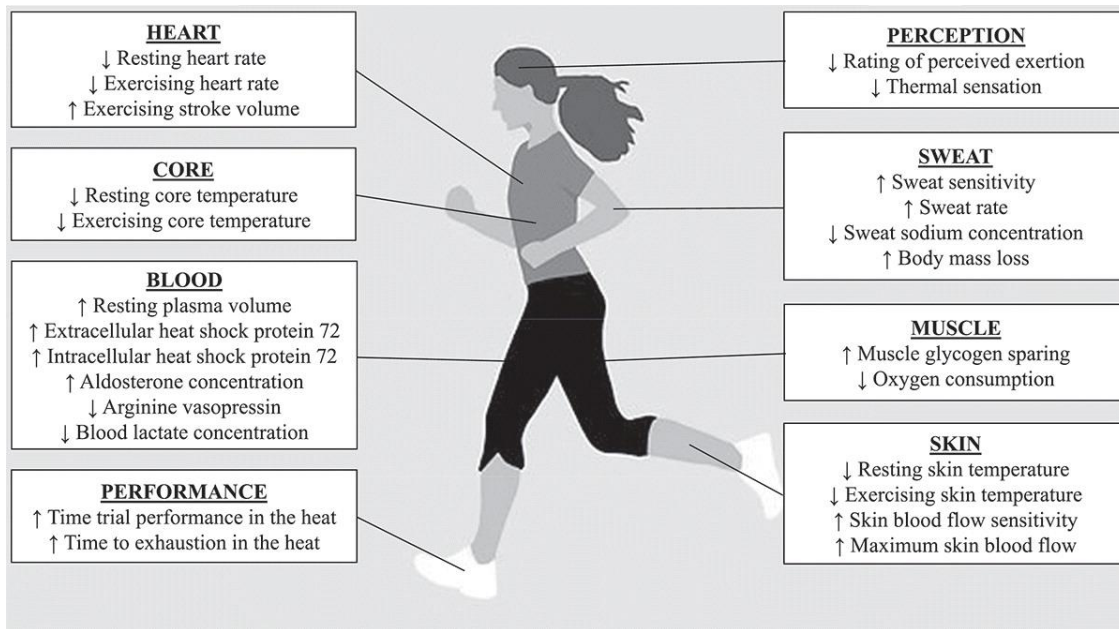


Figure 7. Physiological and Perceptual Adaptations during Heat Acclimation (Pryor *et al.*, 2019a).

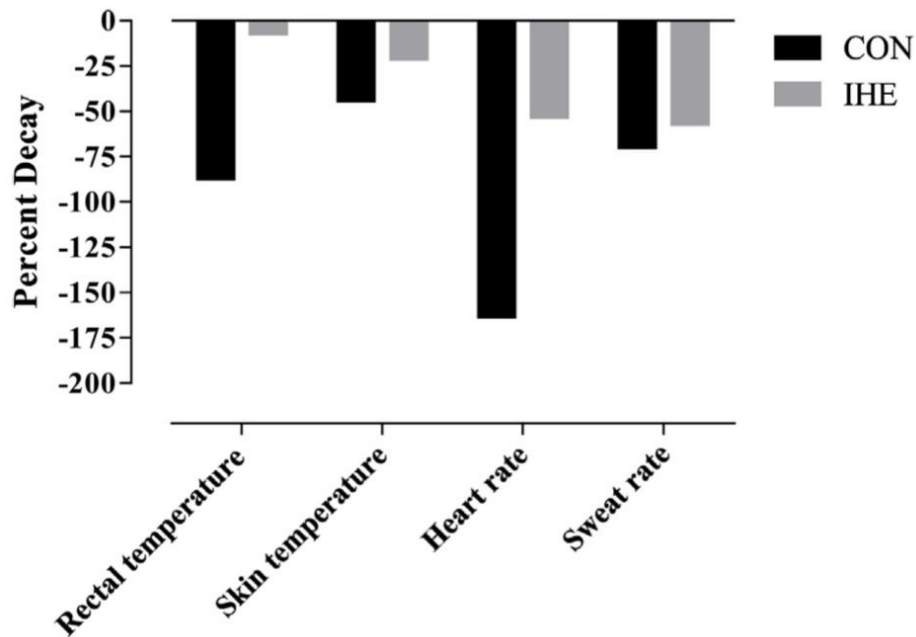


Figure 8. Group comparison of adaptation decay after 25 days removed from the heat (Pryor *et al.*, 2019b).

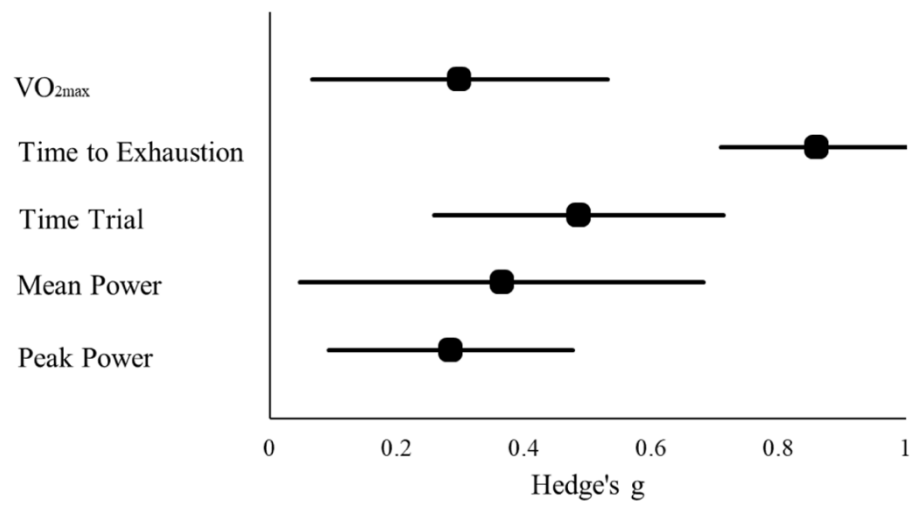


Figure 9. Improvements in Performance following heat acclimation (Benjamin *et al.*, 2019).

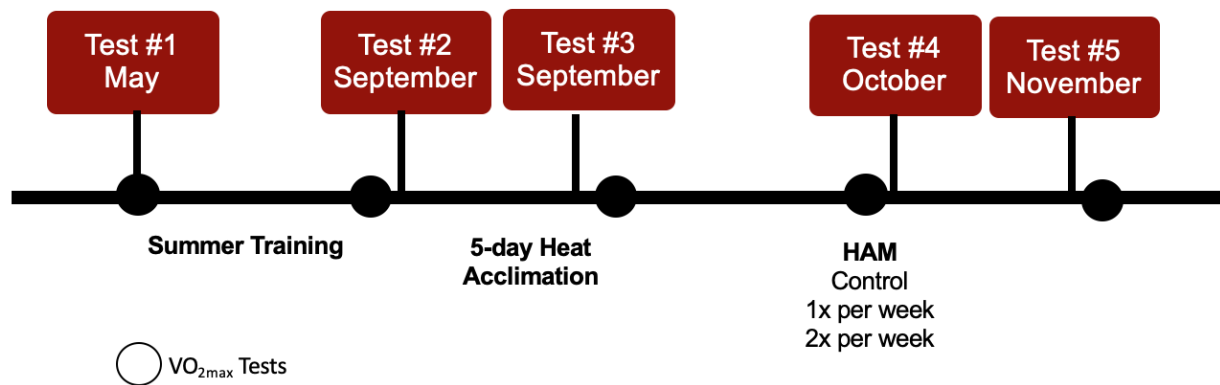


Figure 10. VO_{2max} test timeline.

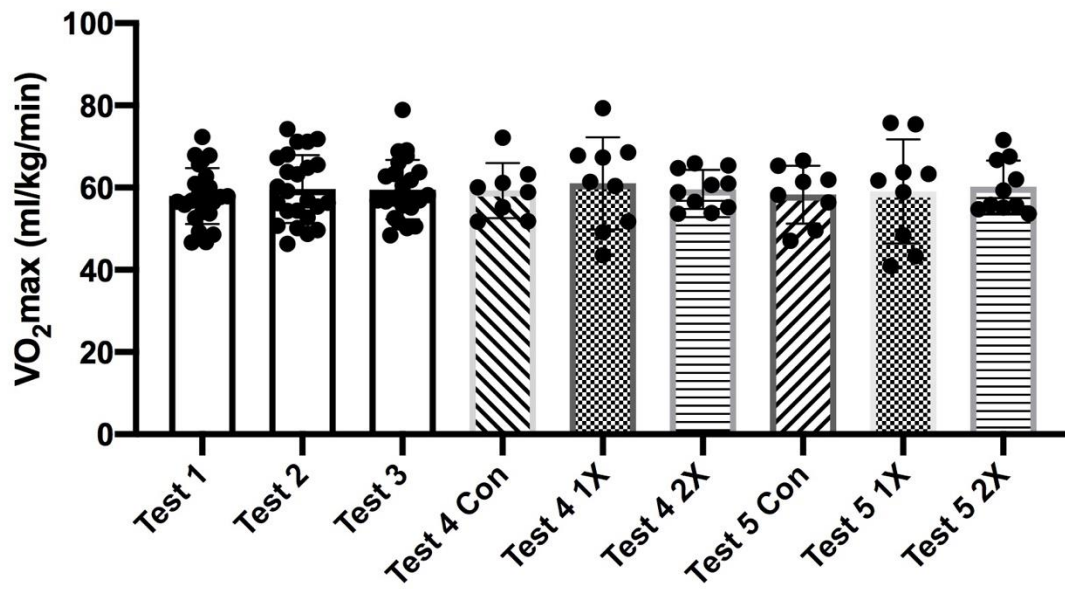


Figure 11. Individual participant VO_2max measurements for Tests 1-3.

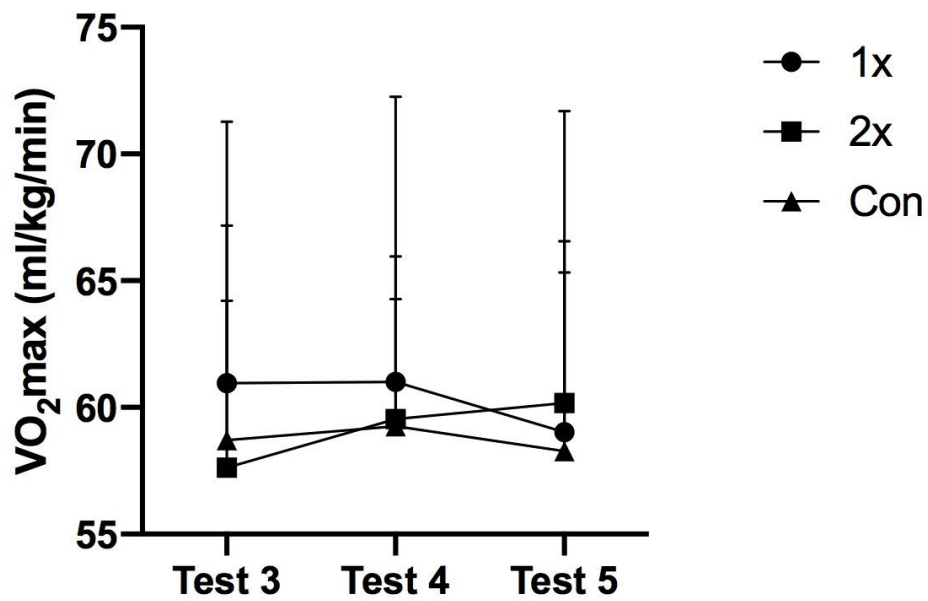


Figure 12. Average VO_2max measurements for Tests 3-5.

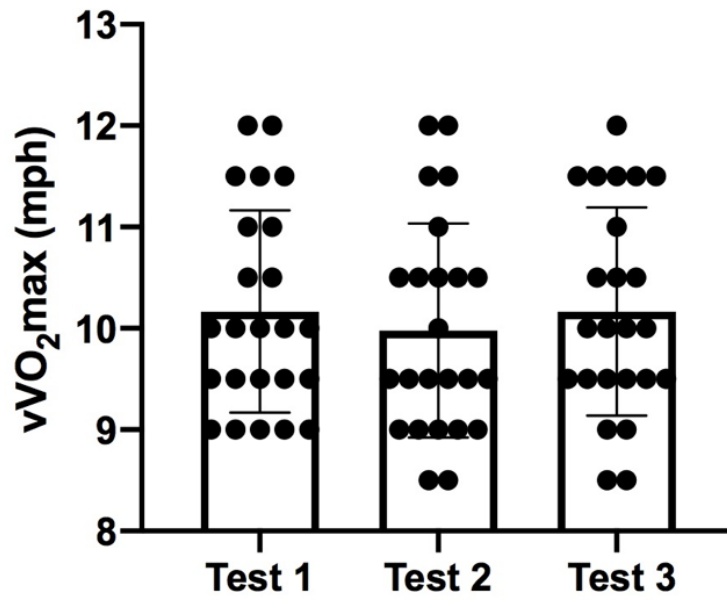


Figure 13. Individual participant $v\text{VO}_2\text{max}$ measurements for Tests 1-3.

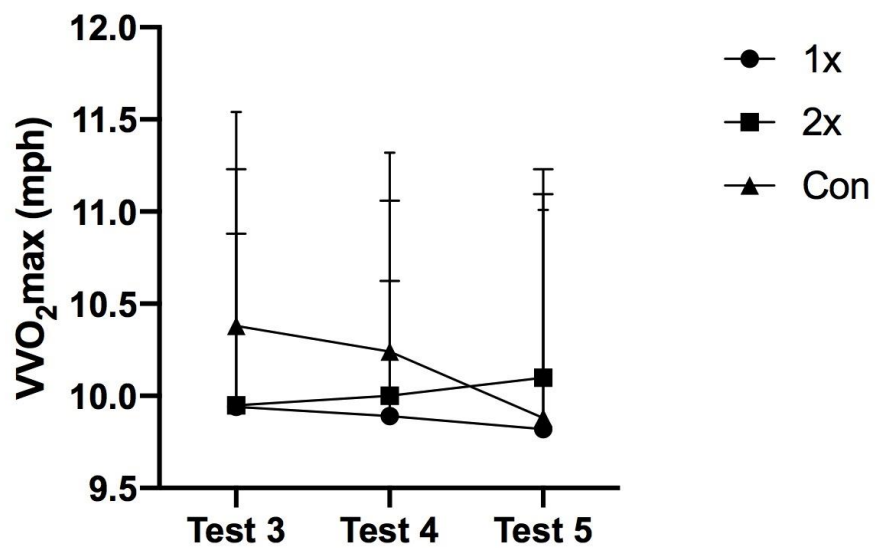


Figure 14. Average $v\text{VO}_2\text{max}$ measurements for Tests 3-5.

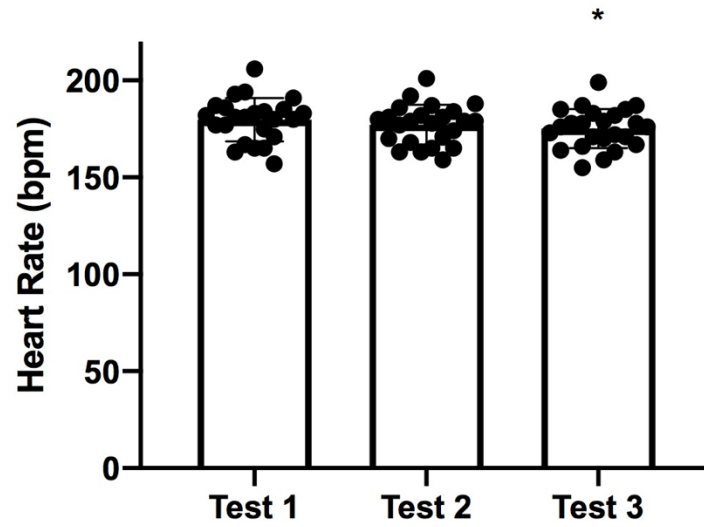


Figure 15. Individual participant maximum heart rate measurements for Tests 1-3.

**Significant from Test 1*

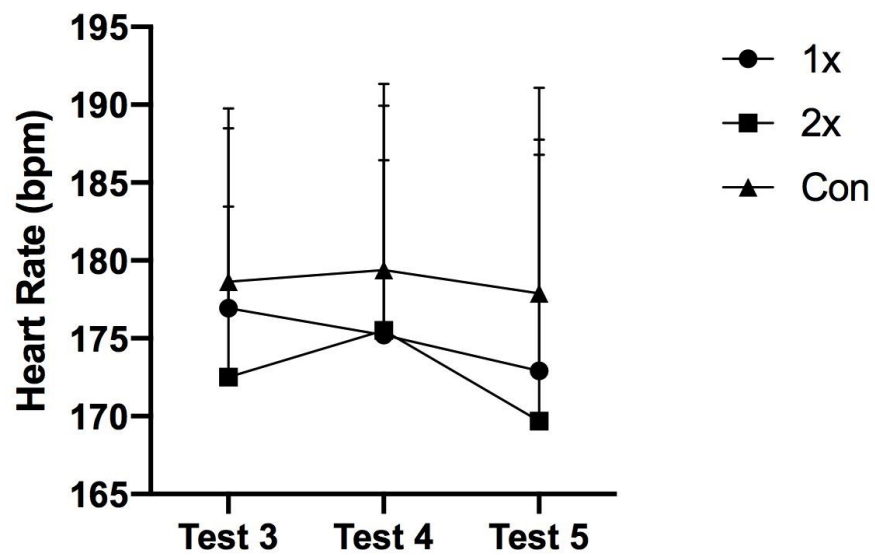


Figure 16. Average maximum heart rate measurements for Tests 3-5.

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